Can J Anesth/J Can Anesth (2011) 58:396–400
DOI 10.1007/s12630-011-9459-9

CASE REPORTS/CASE SERIES

Dynamic hyperinflation and cardiac arrest during one-lung ventilation: a case report

Hyperinflation dynamique et arrêt cardiaque pendant la ventilation sélective: une présentation de cas

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Received: 25 October 2010 / Accepted: 6 January 2011 / Published online: 22 January 2011

Abstract

Purpose *Dynamic hyperinflation describes the phenomenon of progressive gas trapping that occurs in patients with severe airflow obstruction. It is associated with significant hemodynamic instability and may precipitate cardiac arrest. This report describes a case of hemodynamic collapse secondary to dynamic hyperinflation in a patient during one-lung ventilation.*

Clinical features *A 50-yr-old male with a pneumothorax secondary to a ruptured bulla was transferred to the operating room for a left bullectomy. Approximately 30 minutes after initiation of one-lung ventilation in the right lateral decubitus position, sudden ST segment elevation and hypotension occurred, which was refractory to large doses of vasopressor. This culminated in a pulseless electrical activity arrest. The patient was immediately placed supine, disconnected from the ventilator circuit, and resuscitated with chest compressions, fluids, and epinephrine. Auscultation of the right chest revealed no air entry, and needle decompression followed by chest tube insertion in the right chest did not demonstrate any evidence of a pneumothorax. Approximately three to five minutes after the onset of the arrest, the patient's hemodynamics stabilized and there was no evidence of ST elevation. The etiology of the arrest was likely due to dynamic hyperinflation.*

Conclusion *This report highlights the importance of having a high index of suspicion for dynamic hyperinflation and the key to its treatment: disconnection from the ventilator circuit and cessation of mechanical ventilation to allow the lungs to return to functional residual capacity.*

Résumé

Objectif *Le terme d'hyperinflation dynamique fait référence au phénomène de piégeage des gaz qui survient chez les patients souffrant d'obstruction grave des voies aériennes. Cet événement est associé à une importante instabilité hémodynamique et peut mener à un arrêt cardiaque. Ce rapport décrit un cas de collapsus hémodynamique secondaire à une hyperinflation dynamique chez un patient durant une ventilation sélective.*

Éléments cliniques *Un homme de 50 ans présentant un pneumothorax à la suite d'une rupture de bulle a été transféré en salle d'opération pour une bullectomie apicale gauche. Environ 30 min après l'amorce de la ventilation sélective en décubitus latéral droit, une élévation subite du segment ST et une hypotension ont été observées, lesquelles étaient réfractaires à de fortes doses de vasopresseurs. Cet épisode a culminé en un arrêt de l'activité électrique sans pouls. Le patient a immédiatement été couché, déconnecté du circuit de ventilation et réanimé à l'aide de compressions thoraciques, de liquides et d'épinéphrine. L'auscultation du thorax droit n'a pas révélé d'entrée d'air, et la décompression à l'aiguille suivie de l'insertion de la sonde thoracique dans le thorax droit n'a pas montré de pneumothorax. Trois à cinq minutes environ après le début de l'arrêt, le patient est redevenu stable*

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hémodynamiquement et aucun signe d'élévation du segment ST n'était visible. L'étiologie de l'arrêt était probablement due à une hyperinflation dynamique.

Conclusion *Ce rapport souligne l'importance d'un indice de suspicion élevé pour l'hyperinflation dynamique et la clé de son traitement, soit la déconnexion du circuit de ventilation et l'interruption de la ventilation mécanique afin de permettre aux poumons de récupérer leur capacité résiduelle fonctionnelle.*

Dynamic hyperinflation occurs when inspiration commences prior to complete expiration of the preceding breath. The resultant "gas trapping" is of particular concern when mechanically ventilating patients with airflow obstruction.[1–4] In addition to respiratory distress, dynamic hyperinflation is associated with significant hemodynamic instability and may even precipitate cardiac arrest. This phenomenon can be both diagnosed and treated by disconnecting the patient from the ventilator circuit to allow the lungs to decompress over an apneic period. Further management involves adjusting ventilatory settings to maximize expiratory time as well as supportive treatment of any hemodynamic compromise. This report describes an unusual case of cardiovascular collapse secondary to dynamic hyperinflation in a patient during one-lung ventilation (OLV). Written informed consent for this publication was obtained from the patient.

Case presentation

A 50-yr-old male presented to hospital with a three-week history of progressive chest pain and shortness of breath. His medical history consisted of a 30 pack-year smoking history, recently diagnosed chronic obstructive pulmonary disease (COPD), and poorly controlled hypertension. There was no history of ischemic heart disease, and the patient had no functional exercise limitations prior to the current illness. His medication list included budesonide/formoterol and fluticasone/salmeterol inhalers, as well as prednisone. However, all these medications were initiated only five days prior to admission by his family physician. Chest *x-ray* revealed a moderate-sized left pneumothorax. This was most likely caused by the rupture of prominent bullae found in the left apical and basilar areas, as demonstrated by chest computed tomography scan (see Figure). Severe bilateral emphysema and a right apical bulla were also observed. On admission, a left-sided chest tube was inserted and placed on suction.

With no resolution of the pneumothorax after three days, the Thoracic Surgery Service transferred the patient to the operating room for an open left bullectomy. On arrival, his vital signs included a heart rate (HR) of 100 beat min^{-1}, blood pressure (BP) of 160/95 mmHg, and an oxygen (O_2) saturation of 94% on 2 $L{\cdot}min^{-1}$ delivered via nasal prongs. The patient's airway exam was unremarkable and tracheal intubation was predicted to be easy. His cardiac exam revealed normal heart sounds with no evidence of murmurs. Chest auscultation demonstrated decreased air entry bilaterally, which was notably worse on the left pneumothorax side. Baseline blood work, including a complete blood count, electrolytes, creatinine, international normalized ratio, and partial thromboplastin time, were all within normal limits. The baseline electrocardiogram was unremarkable.

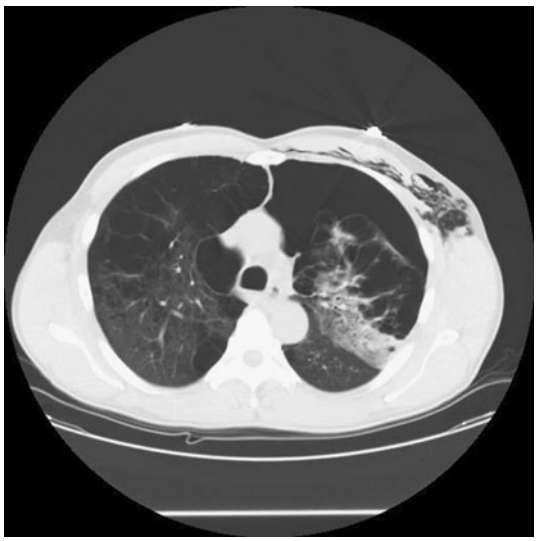

Figure Thoracic computed tomography (CT) scan of patient demonstrating bilateral bullae, emphysema, and left-sided pneumothorax

Standard monitors were applied in the operating room. Prior to induction, a right radial arterial line was placed and an epidural was inserted at the T6-7 interspace, both without complication. In addition, the epidural was tested with 3 mL of 2% lidocaine prior to induction. The patient was then induced with midazolam 2 mg *iv*, fentanyl 250 μg *iv*, lidocaine 30 mg *iv*, propofol 200 mg *iv*, and rocuronium 65 mg *iv*. A size 41 French left-sided double lumen tube (DLT) was inserted easily. Following confirmation of DLT placement with fibreoptic bronchoscopy, right-sided OLV was initiated. After placing the patient in the right lateral decubitus position, the placement of the DLT was re-confirmed by bronchoscopy. To minimize the risk of rupturing the right sided bulla and causing a pneumothorax, a ventilation strategy focused on maintaining low airway pressures was used. Initially, the right lung was ventilated using volume-controlled ventilation with a tidal volume of 400 mL, a respiratory rate of 14 breaths$\cdot min^{-1}$, a fractional inspired oxygen concentration of 1.0, and an inspired to

expired (I:E) ratio of 1:2. Maintenance of anesthesia was achieved with sevoflurane. With these ventilatory parameters, the patient's vital signs were: HR = 100 beats·min^{-1}, BP = 110/60 mmHg, and O_2 saturation = 98%. An upsloping capnography trace and an end-tidal carbon dioxide of 35 mmHg were noted. The resulting peak airway pressure was 23 cm of water (cm H_2O).

Approximately 30 minutes after the onset of OLV, ST segment elevation of approximately 3 mm was noted in leads II and V5. This was associated with an increase in the patient's HR to 115 beats·min^{-1} and a decrease in BP to 80/50 mmHg. At the same time, an increase in the peak airway pressure to 30 cm H_2O was also noted. Oxygen saturation remained at 98%. Multiple boluses of phenylephrine totalling 360 μg were given intravenously to treat the hypotension. Once the systolic BP was > 100 mmHg, esmolol 5 mg *iv* was administered cautiously to decrease the HR in an attempt to improve myocardial perfusion and reduce oxygen demand. Bronchoscopy revealed that the tracheal lumen was positioned slightly below the carina, and the DLT was repositioned. However, this maneuver did not lead to any change in the peak airway pressures. Though the BP responded initially to phenylephrine administration, hypotension reoccurred over the next five minutes and became refractory to further boluses of phenylephrine totalling 800 μg *iv*. This episode culminated in a pulseless electrical activity (PEA) arrest. Of note, the oxygen saturation remained at 98% through the initial resuscitation up to the onset of PEA arrest. The patient was immediately placed supine, and resuscitation was initiated according to the Advanced Cardiac Life Support protocol. Manual hand bag ventilation was not associated with significant resistance. Auscultation over the patient's right chest revealed minimal breath sounds.

Based on the patient's clinical history of COPD and bullous lung disease, the elevated peak airway pressures, and the lack of right-sided air entry, our main differential diagnosis at this time was tension pneumothorax and dynamic hyperinflation. A consequence of both differentials would include cardiac outflow obstruction and cardiac ischemia in multiple coronary territories. Although less likely, an acute thrombotic event that would result in ST elevation in two non-contiguous leads could not be ruled out at the time of this critical event. A large pulmonary embolus (e.g., thrombus or air) was also considered; however, oxygen desaturation would have been expected, and an increase in airway pressures would be unusual. Our differential diagnosis also included a profound neuraxial block. However, this seemed unlikely since the onset of hypotension was more than 60 min after the epidural test dose was administered. Although anaphylaxis would have accounted for the noted hemodynamic changes and the increase in airway pressure, we placed this possibility lower on our differential because lung compliance on hand bag ventilation was not noticeably low, and more than 45 min had elapsed since the last administration of intravenous medication. At the time of this event, there was no evidence of massive blood loss.

Given the high index of suspicion for a tension pneumothorax and dynamic hyperinflation, the endotracheal tube was disconnected from the ventilator circuit at the onset of the PEA arrest. Chest compressions were initiated and the patient was resuscitated with a fluid bolus of 1 L, epinephrine 1 mg *iv*, and calcium chloride 1 g *iv*. Approximately two minutes after the administration of these drugs, vasopression 40 U *iv* was also given. A right-sided needle decompression was performed, followed by chest tube insertion. A tension pneumothorax was ruled out because neither intervention was associated with the release of pressurized air or any clinical improvement. In addition, no bubbling was noted in the chest tube water seal device. After three to five minutes of resuscitation, the patient suddenly and dramatically converted from PEA to sinus rhythm with a HR of 130 beats·min^{-1} and a BP of 240/125. Nitroglycerine 40 μg *iv* was given to manage the post-resuscitation hypertension.

The only electrocardiogram abnormality following the resuscitation was T-wave inversion. An arterial blood gas preformed immediately after the arrest with the patient still on OLV, and an F_IO_2 of 1.0 showed a pH of 7.16, pCO_2 of 66, pO_2 of 148, and HCO_3 of 24; electrolytes and hemoglobin were within normal limits.

The sudden resolution of the arrest followed by extreme hypertension strongly suggested that the underlying cause was cardiac outflow obstruction. Once the obstruction was relieved and circulation was re-established, the resuscitative drugs entered the circulation, which resulted in significant hypertension. Having ruled out a right-sided tension pneumothorax, dynamic hyperinflation and pulmonary embolus were the two remaining causes of obstructive shock from our differential diagnosis. Intraoperative transesophageal echocardiography was unavailable to assess definitively whether a pulmonary embolus was present. However, evidence of unimpaired oxygenation immediately following the arrest would argue against this diagnosis.

With dynamic hyperinflation remaining high on our differential, the respiratory rate and the I:E ratio were decreased to 8 breaths·min^{-1} and 1:4.5, respectively, in order to facilitate adequate expiration. The patient was also switched to pressure-controlled ventilation of 24 cm H_2O, which resulted in tidal volumes of 450 to 500 mL. With these changes, the patient was hemodynamically stable for the remainder of the bullectomy with a HR of 100 to 110 beats·min^{-1} and a systolic BP of 100 to 130 mmHg. Following the resuscitation, no further use of vasopressors or

inotropes was needed. At the conclusion of the case, the DLT was exchanged for a single lumen endotracheal tube. To facilitate a controlled emergence on pressure support ventilation and to provide further optimization with inhaled bronchodilator therapy, the patient was brought to the postanesthetic care unit intubated. He was extubated three hours later, and then transferred to the Thoracic Step-down Unit.

Postoperatively, the patient's troponin level peaked at 0.15 $\mu g \cdot L^{-1}$ and then normalized within 24 hr. A transthoracic echocardiogram in the postanesthetic care unit showed normal right and left ventricular function and no major wall motion abnormalities. These findings were helpful in excluding coronary artery disease as a cause for the PEA arrest. The Cardiology Service followed the patient and concluded that his subsequent electrocardiograms showed no evidence of myocardial ischemia or infarction.

On postoperative day 12, the patient was discharged home with a chest tube in place. However, he subsequently developed a left-sided empyema and *Staphylococcus aureus bacteremia* that required re-hospitalization for intravenous antibiotics and chest tube maintenance.

Discussion

This case report demonstrates an example of dynamic hyperinflation during OLV that caused profound hemodynamic instability and collapse. Dynamic hyperinflation during thoracic surgery was first described in patients suffering from end-stage emphysema due to alpha-1 antitrypsin deficiency who were undergoing lung transplantation.[2] It can occur in patients with airflow obstruction, such as in COPD, asthma, and bronchiectasis, as well as in patients with central airway obstruction from tumours or foreign bodies.[4]

Dynamic hyperinflation occurs when there is insufficient time for complete exhalation before the next breath is initiated. Factors that contribute to the development of dynamic hyperinflation include a large tidal volume, short expiratory time, and a high degree of airway obstruction. This leads to breath-stacking and a buildup of intrinsic positive end-expiratory pressure (PEEP).[2,5] Distended alveoli compress pulmonary capillaries, which increases dead space and leads to inefficient gas exchange.[4] From a cardiovascular perspective, the increased intrathoracic pressure leads to decreased venous return to the heart in a way that is akin to cardiac tamponade.[1,3,4] Increased pulmonary vascular resistance as well as high right-ventricular afterload associated with dynamic hyperinflation also contribute to a reduction in left-ventricular compliance and contractility and therefore a drop in cardiac output.[2] This can result in hemodynamic collapse and cardiac arrest that presents most commonly in the form of PEA.

Most reports of dynamic hyperinflation in the literature involve late recognition and prolonged hemodynamic depression as a result.[1,2] Indeed, there have been case reports of dynamic hyperinflation being diagnosed only after the resuscitation has been stopped, the patient pronounced dead, and the ventilator turned off.[6,7] Early signs of dynamic hyperinflation include hypotension, increased jugular venous distension, pulsus paradoxus on the arterial waveform, and oxygen desaturation.[2,4] Many reports have described different methods of detecting or quantifying the amount of hyperinflation: collecting expired gas into a ventilator bellow; observing increased peak and plateau airway pressures during volume-controlled ventilation; observing decreased tidal volumes during pressure-controlled ventilation; measuring the amount of intrinsic PEEP; and observing persistent expiratory flow at the end of expiration on a flow-volume loop. Most of these methods, however, are fraught with inherent complications that lead to inaccurate measurement. Consequently, no clear method is recommended to monitor for dynamic hyperinflation. A high level of suspicion based on the clinical scenario is crucial.

Prevention of dynamic hyperinflation is based on the underlying determinants of the problem: ventilator manipulation to optimize tidal volume and expiratory time, and pharmacologic therapy to reverse airway obstruction. General principles of ventilation include using a low tidal volume, low respiratory rate, and an I:E ratio that prolongs expiration (see Table). In several studies, different absolute values for these parameters have been recommended, but the general principles remain constant.[1–4,8] A degree of hypoventilation and therefore hypercarbia is accepted. Based on the current literature, the use of PEEP is controversial. The splinting effect of PEEP maintains airway patency throughout the respiratory cycle, which is advantageous in patients with small airway collapse and may minimize the amount of trapped air.[4,5,8] However, given the potential that extrinsic PEEP may increase total PEEP and the inherent difficulty in measuring total PEEP

Table Recommended ventilator settings to reduce dynamic hyperinflation

Tidal volume	6 to 8 $mL \cdot kg^{-1}$
Respiratory rate	4 to 10 $breaths \cdot min^{-1}$
I:E ratio	$\leq$1:3
F_IO_2	Maintain SpO_2 >90%
Inspiratory flow rate	60 to 100 $L \cdot min^{-1}$
Plateau pressure	<30 cm H_2O

I:E = inspired to expired

intraoperatively, our institution does not promote the addition of extrinsic PEEP in patients with severe airflow obstruction.

Suspected dynamic hyperinflation is both diagnosed and managed by the apnea test, i.e., the patient is disconnected from the ventilator and the lungs are allowed to return to functional residual capacity (FRC).[3,4,9,10] Circuit disconnection and apnea may be needed for up to several minutes, as the amount of gas trapped can exceed several litres.[2] Other methods of treatment include the use of bronchodilating agents (beta agonists, corticosteroids, volatile anesthetics), fluid resuscitation to optimize preload, and inotropic drugs.[2,4] However, as in our case report, inotropic drugs administered during a resuscitation may not enter the circulation until the dynamic hyperinflation resolves, resulting in a delayed effect and profound hypertension. In one such case, this combination of factors precipitated an intracranial hemorrhage and the patient's subsequent death.[1]

When dynamic hyperinflation results in an arrest, the performance of chest compressions during resuscitation can also assist in decompressing the lungs.

Surgical maneuvers include prompt entry into the thoracic cavity and manual decompression of the lungs. The use of cardiopulmonary bypass has been described in cases of prolonged hemodynamic collapse refractory to resuscitation.[2]

In conclusion, dynamic hyperinflation during surgery in patients with obstructive lung disease is an underrecognized phenomenon that can cause hemodynamic instability or intraoperative arrest. This case report highlights the importance of having a high index of suspicion for dynamic hyperinflation. In patients with severe obstructive lung disease, the use of ventilation strategies that optimize expiratory time can minimize the risk of dynamic hyperinflation. To manage and treat dynamic hyperinflation, patients whose lungs are mechanically ventilated should be disconnected from the breathing circuit to allow the lungs to return to FRC. Though this measure is not part of standard resuscitation protocols, it is crucial for the diagnosis and treatment of this complication.

Competing interests None declared.

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